Non-fasting Non-High Density Lipoprotein Cholesterol (Non-HDL-C) AS A predictor of Atherosclerosis in Patients with End Stage Renal Disease on Regular Hemodialysis

Sabry Gohar*, Mone Hosny* Howayda Abdel-Hamid*, and Yasser Ibrahim**

* Internal Medicine Department, Ain Shams University

**Damanhour Medical Institute

Abstract

Fifty patients with chronic renal failure on regular hemodialysis 31 males and 19 females, aged from 18 to 62 years were included in the study. They were selected from those attending the Nephrology and Dialysis Unit in Damanhour Medical Institute.

Ten age and sex matched subjects with normal renal function served as control group. Full history taking, clinical examination, resting ECG and serum cholesterol, HDL (non-fasting), fasting LDL, non-fasting non-HDL from special equation, Non- HDL = total cholesterol - HDL and Duplex scanning of both carotid and femoral arteries.

Non fasting non-HDL was more than 130 mg/dl in more than 65% of patients group versus 30% in control group, serum level of non-fasting cholesterol and non-fasting non-HDL were significantly elevated in hemodialysis patients as compared with control group, while serum level of non-fasting HDL and fasting LDL were significantly decreased in hemodialysis patients as compared with control group. Intima-Media thickness (IMT) of both carotids and both femoral arteries was elevated in hemodialysis patients as compared with control group. There was a significant positive correlation between non-fasting non-HDL and IMT of both carotids and both femoral arteries and a significant positive correlation between fasting LDL and IMT of both carotids and femoral arteries. There was a significant positive correlation between duration of dialysis and IMT of both carotids and left femoral arteries.

A positive correlation exists between ECG ischemic changes and IMT of both carotids and left femoral arteries and between carotid plaques and IMT of both carotids and left femoral arteries.

Aim of the Work

To study and compare between the level of non-fasting non-HDL-C and fasting LDL in predialysis serum as predictor of atherosclerotic changes in patients with end stage renal disease on regular hemodialysis.

Indroduction

ESRD patients show various abnormalities in plasma lipids and lipoproteins that are called uremic dyslipidemia (Shoji *et al.*, 1997).

Atherosclerosis and cardiovascular disturbance are common among patients

with progressive renal insufficiency and in uremic patients receiving long-term hemodialysis (Jungers *et al.*, 1997).

Cardiovascular disease is the most important cause of mortality in end-stage renal disease (Merzog *et al.*, 1999).

Plasma lipid disturbance have been identified as significant risk factor for cardiovascular disease in end-stage renal disease patients (Mittman *et al.*, 1996).

The main lipid abnormality is an increase in plasma triglyceride and a decrease in HDL cholesterol concentrations with smaller change in the levels of cholesterol rich lipoproteins (Koniger *et al.*, 1999).

Lipoprotein changes have been shown to be closely associated with atherosclerosis in ESRD patients. Non-HDL cholesterol (cholesterol in VLDL + IDL + LDL fraction) was shown to be an independent factor for both carotid artery intima-media thickness (Shoji *et al.*, 2000).

Patients and Methods

50 patients with chronic renal failure on regular hemodialysis, 31 males and 19 females, with age ranging from 18 to 62 years, were included in the study. They were selected from those attending the nephrology and dialysis unit in Damanhour Medical Institute.

Dialysis sessions were done 3 times weekly, each session 4 hours on bicarbonate dialysate, with biocompatible membrane (polysulphone).

Ten age and sex matched subjects with normal renal functions (7 males and 3 females), aged from 22 to 63 years served as a control group; they were selected from those attending the sonography departement We excluded from the study patients with obesity, diabetes mellitus, liver disease, or other metabolic or endocrine disorders and those receiving drugs known to alter lipid or lipoprotein patterns such as duiretics, β -blocker were excluded.

Both the control and patients groups were subjected to the following:

1.Full history taking as regards the original disease of renal failure, ischemic symptoms (cerebral, coronary and peripheral).

- 2.Thorough clinical examination including CNS, CVS and peripheral vessels.
- 3.Standard resting electrocardiograms (ECG).
- 4.Lipid studies including LDL (14 hours fasting), total cholesterol and high density lipoproteins (non-fasting).

Blood samples were taken at the start of hemodialysis session. Total cholesterol was assayed enzymatically and HDL by precipitation method with dextran sulphate and magnesium chloride.

Non-fasting non-HDL from special equation:

Non-HDL = total cholesterol - HDL.

 Duplex scanning of carotid and femoral arteries by general electronic volusion 750 using 7.5 MHz high resolution linear array transducer in the supine position with slight hyperextension of the neck.

Identification of carotid arteries was performed in both transverse longitudinal sections by placing transducer medial to the sternomastoid muscle. Optimal measurement of the intima-media thickness was obtained along the course of common carotid artery 2 cm proximal to the bulb after grey scale magnification to obtain detailed structure of the carotid wall. Identification of femoral arteries in the supine position, just under the middle of inguinal ligament.

Results

Aetiology of renal failure was as follows: glomerulonephritis in 14 patients out of 50(28%), hypertension in 17 patients out of 50 (34%), pyelonephritis in 5 patients out of 50 (10%), polycystic kidney in 2 patients out of 50(4%), analgesic nephropathy in 2 patients out of 50 (4%) and unknown in 10 patients (20%).

Table (1): Demographic data of the patients (n=50) and controls (n=10).

	Patients (n =50)	Control (n=10)	
Sex:			
¥ Male.	31(62.0%)	7(70.0%)	
¥ Female	19(38.0%)	3(30.0%)	
X2	0.23		
P value	0.63 (non-significant)		
Age (in years):			
¥ Range.	18-62	22-63	
¥ Mean±SD	40.4±11.88	41.9±13.33	
T	0.41		
P value	0.68 (non-significant)		

Table (2): Distribution of patients group (n=50) according to duration of dialysis.

	Frequency		
	No (out of 50)	%	
< 12 months	7	14.0%	
> 12- ² 36 months	18	36.0%	
> 36- ² 60 months	19	38.0%	
More than 60 months	6 12.0		
Range (months)	3-132		
Mean ±SD	38.28±24.63		

Table (3): Comparative study as regards serum lipid profile between patients under regular hemodialysis and control group.

Lipid profile	Patients (n=50)	Control (n=10)	t, p value
Non fasting cholesterol (in			
mg/dl)			
Range	141-308	150-200	4.65
Mean ±SD	200.5±37.43	169.1±16.81	0.003* H.S.
Non fasting HDlL (in mg/dl)			
Range	28-64	35-66	1.12
Mean±SD	45.52±8.35	48.8±10.01	0.1757 N.S.
Non-fasting non-HDL (in mg/dl)			
Range			
Mean±SD	91-280	84.143	4.21
	151.2±39.68	110±38.44	0.0044* H.S
Fasting LDL (in mg/dl)	_		
Range.	62-174	88-144	1.03
Mean±S.D	107.52±26.07	115.5±17.77	0.1253 N.S.

Table (4): Comparaison between the patients group (n=50) and control group (n=10) regarding intima-media thickness (IMT) of both carotids and both femoral arteries thickness.

Lipid profile	Patients (n=50)	Control (n=10)	t, p value
IMT left carotid (in cm)			
Range	0.073-0.171	0.065-0.110	3.21
Mean ±SD	0.122 ± 0.035	0.0883±0.021	0.01* H.S.
IMT right carotid (in cm)			
Range	0.080-0.175	0.052-0104	2.85
Mean±SD	0.137 ± 0.13	0.078±0.012	0.031 S.
IMT left femoral (in cm)			
Range	0.075-0.1777	0.052-0.104	2.85
Mean±SD	0.126 ± 0.034	0.078±0.012	0.026* S.
IMT of right femoral (in cm)			
Range.	0.075-0.166	0.088-0.106	6.21
Mean±S.D	0.122±0.022	0.0987±0.01	0.0001* H.S.

¥ IMT: intima-media thickness.

Table (5): Correlation between lipid profile and intima-media thickness (IMT) of both carotid and both femoral arteries in patients group.

Cholesterol		HDL	Non HDL	LDL
IMT of left carotid				
r	0.10	0.01	0.45	0.40
P	>0.05 N.S.	>0.05 N.S.	<0.05* S.	<0.05* S.
IMT of right carotid				
r	0.29	0.29	0.48	0.43
P	>0.05 N.S	>0.05 N.S.	<0.05* S	<0.05* S.
IMT of left femoral				
r	-0.23	-0.11	-0.35	-0.34
P	>0.05 N.S.	>0.05 N.S.	<0.05* S.	<0.05* S.
IMT of right femoral				
r	-0.22	-0.03	-0.37	-0.30
P	>0.05 N.S.	>0.05 N.S.	<0.05* S.	<0.05* S.

Table (6): Distribution of ECG ischaemic changes and carotid plaques in patients group (n=50).

	Freque	ency
	No. out of 50	%
ECG ischaemic changes		
+ve	8	16.0%
-ve	42	84.0%
Carotid plaques		
+ve	7	14.0%
-ve	43	86.0%

Table (7):Comparaison between lipid profile and ECG findings in patients group (n=50), group 1.

ECG finding	Patients with -ve	Patients with +ve	t, P value
	finding of ECG	finding of ECG	
Lipid profile (mg/dl)			
non fasting cholesterol (mg/dl)			
Range.	141-290	180-308	2.45
Mean±SD	190.33±41.62	226.25±46.570	0.018** H.S.
Non fasting HDL (mg/dl):			
Range.	30-64	28-54	1.65
Mean±SD	45.81±8.36	44 ± 8.668	0.32 N.S.
Non fasting non HDL (mg/dl)			
Range.	91-256	143-280	2.98
Mean±SD	145.05 ± 35.10	183.5 ± 48.858	0.02* S.
Fasting LDL (mg/dl)			
Range.	62-174	85-174	1.89
Mean±SD	105.00±25.21	120.75±28.238	0.04* S,

Table (8): Comparaison between ECG findings and (IMT) of both carotids and both femoral arteries in patients group (n=50).

	ECG finding	Patients with -ve finding of ECG	Patients with +ve finding of ECG	t, P value
IMT (cm)		imang of Doo	initially of 200	
IMT of left carotid				
Range.		0.073-0.171	0.099-0.162	1.99
Mean±SD		0.110 ± 0.04	0.135 ± 0.022	0.045* S.
IMT of right carotid				
Range.		0.075-0.175	0.085-0.177	4.15
Mean±SD		0.083 ± 0.03	0.136 ± 0.022	0.008** H.S
IMT of left femoral				
Range.		0.07-0.171	0.085-0.177	3.82
Mean±SD		0.091 ± 0.009	0.116±0.027	0.009** H.S.
IMT of right femoral				
Range.		0.075-0.166	0.089-0.144	1.05
Mean±SD		0.112±0.02	0.116±0.016	0.187 N.S.

Table (9): Comparaison between lipid profile and carotid plaques in patients group (n =50).

Carotid plaques	Patients with -ve	Patients with +ve	t, P value
	finding of carotid	finding of carotid	
Lipid profile (mg/dl)	plaques	plaques	
Cholesterol			
Range.	141-308	193-245	2.34
Mean±SD	200.36±50.93	222.43±25.17	0.032* S.
HDL			
Range.	28-64	33-62	0.98
Mean±SD	46.60±8.38	44.71±9.32	0.34 N.S.
Non HDL			
Range .	91-280	130-194	2.65
Mean±SD	142.26±42.54	167.71 ± 20.71	0.01* S.
LDL			
Range.	62-174	83-125	1.03
Mean±SD	107.57±27.66	103.71±14.40	0.21 N.S.

Table (10): Comparaison between carotid plaques and IMT findings in patients group (n=50).

Carotid plaqu		Patients with +ve	t, P
	finding of carotid	finding of carotid	value
IMT (cm)	plaques	plaques	
IMT of left carotid			
Range.	0.073-0.109	0.073-0.171	3.21
Mean±SD	0.142 ± 0.05	0.101±0.032	0.02* S.
IMT of right carotid			
Range.	0.080-0.175	0.083-0.160	1.98
Mean±SD	0.124 ± 0.03	0.110 ± 0.02	0.042* S.
IMT of left femoral			
Range.	0.075-0.177	0.073-0.149	1.99
Mean±SD	0.141 ± 0.041	0.111±0.02	0.041*S.
IMT of right femoral			
Range.	0.075-0.166	0.083-0.149	0.98
Mean±SD	0.112±0.02	0.112±0.02	0.41 N.S.

Table (11): Correlation between duration of dialysis and intima-media thickness (IMT) of both carotids and femoral arteries.

	Duration of dialysis	
	r	P
IMT of left carotid	0.398	0.012* S.
IMT of right carotid	0.42	0.003** H.S.
IMT of left femoral	0.61	0.0001 H.S.
IMT of right femoral	0.21	0.31 N.S.

Discussion

Cardiovascular disease is the major cause of death in ESRD patients (U.S. Renal Data System, 2003). Dialysis patie-nts have a cardiovascular mortality rate that is 10-20 times higher than the general population (Sarnak *et al.*, 2000).

There are many reasons why CKD is associated with CVD. In cross sectional studies, individuals with CKD have more severe atherosclerosis(Shlipak *et al.*, 2002).

A percentage of 30% of hemodialysis patients clinically have ischemic episodes with normal coronary angiograms (Rostand *et al.*, 1984).

Hemodialysis patients with cardiovascular disease exhibited higher levels of triglycerides (Koch *et al.*, 1997), VLDL-cholesterol (Mahn *et al.*, 1983), total cholesterol, LDL-cholesterol and lower levels of HDL-cholesterol and lower levels of HDL cholesterol (Koch *et al.*, 1997) than those without cardiovascular disorders.

It has also been found that hemodialysis patients with a rapid increase in total amount of coronary artery calcification have higher triglyceride levels and lower HDL-cholesterol levels compared with patients who exhibit slow progress of coronary vessel lesions (Takashiro *et al.*, 2001).

This study included fifty patients with chronic renal failure on regular hemodialysis 31 males and 19 females aged from 18 to 62 years with a (mean ±SD) age of (41.9±13.35) years served as control group.

Official recommendations in the general population employ LDL in fasting blood as gold standard lipid parameter in lipid related risk assessment. There are some problems in the use of fasting LDL-C among ESRD patients treated by hemodialysis. First, the use of only LDL-C ignores the atherogenic potentials of triglyceride-rich lipoproteins. The degree of

association of IDL and VLDL with aortic sclerosis was greater than that of LDL in hemodialysis patients (Shoji et al., 1998). Second, it is difficult for many patients to obtain blood samples after overnight fasting for standard lipid analysis. Because plasma triglyceride level is greatly affected by eating, the friedural formula, gives falsely lower LDL-C if non-fasting specimens are used. More recent. National cholesterol education program adult treatment panel III (NCEP ATP-III), Executive summary of the third report of the National Cholesterol Education Program (NCEP, 2001), listed non-HDL-C, as well as LDL-C, as target lipid parameters in subject hypertriglyceridemia.

Non-HDL-C is the sum of LDL-C and choleserol in triglyceride rich lipoproteins, but can be easily calculated by subtracting HDL-C from total cholesterol. Because HDL-C is hardly affected by eating, (Wilder *et al.* 1995), non HDL-C may be a better index than LDL-C for routine practice in most of hemodialysis patients. Non fasting non-HDL-C can be easily calculated from total cholesterol and HDL-C without limitation in hypertriglyceridemia.

In this study, (table 3), the serum level of cholesterol and non-fasting non-HDL were significantly elevated in hemodialysis patients compared to control group (P = 0.003 and P=0.044), respectively. This goes with (Attman *et al.*, 1991), who found that dialysis patients with vascular disease have increases in serum triglycerides, total cholesterol and VLDL cholesterol with lower levels of HDL-cholesterol.

Non-fasting non-HDL-C is a marker that integrates atherogenic potentials carried by VLDL, IDL and LDL. In the study of Shoji et al. (1998), although IDL was the lipoprotein that was most closely associated with aortic sclerosis, VLDL and LDL were also significantly associated with aortic sclerosis independent of other nonlipid variables. Therefore, use of only one lipoprotein level may miss the significant contribution other lipoproteins of Nishizawa et al. (2003) has evaluated the power of non-fasting non-HDL-C predialysis serum as a predictor cardiovascular mortality in a cohort of 525

hemodialysis patients. During the mean follow-up of 64 months, 120 deaths, including 44 fatal cardiovascular events, occurred. Patients in the highest tertile of non-fasting non-HDL-C (137 to 285 mg/dl) had a significantly higher risk for cardiovascular mortality.

They concluded that non-fasting non-HDL in predialysis serum was a significant and independent predictor of cardiovascular mortality in hemodialysis patients.

The early stages of atherosclerosis are associated with changes in arterial structure. Subtle structural changes such as thickening of IMT occur early in the atherosclerotic disease process (Bernadette et al., 2001; Kato et al., 2003 and Papagiani et al., 2003). Ultrasonic evaluation of carotid artery for IMT of carotid and femoral arteries (superficial vascular districts) can identify patients at risk for cardiovascular disease.

In this study, (table 4), IMT of both carotids and both femoral arteries were elevated in hemodialysis patients as compared to control group. This goes with most studies that found a significant increase in IMT in the carotid arteries of HD patients compared with healthy control subjects (Kawagishi *et al.*, 1995; London *et al.*, 1996 and Mojs *et al.*, 2000).

Benedetto *et al.* (2001) found that IMT may be usefully applied for risk stratification in the dialysis population. Damjavonic *et al.* (2003) evaluated IMT of 45 dialysis patients and found higher mean carotid IMT in HD patients than in control group Kagaguishi *et al.* (1995), showed that IMT was significantly higher in HD patients than in age and gender matched control subjects.

In this study, there was no significant correlation between level of non-fasting serum cholesterol, non-fasting HDL and IMT of both carotids and both femoral arteries (table 5), while there was a significant positive correlation between non-fasting non-HDL and IMT of both carotids and both femoral arteries. Also, there was a significant positive correlation between fasting LDL and IMT of both carotids and both femoral arteries (P<0.05).

This goes with most results of Kawagishi et al. (1995); London et al., 1996), that failed to find a relationship between IMT and serum cholesterol and triglycerides in dialysis patients, probably relationship because the of cholesterol to mortality is U-shaped (Lowrie et al., 1990). Increased mortality risk at low serum cholesterol levels most likely reflects confounding malnutrition in these patients. Sarnak et al. (2000) and Burdick et al. (1994) found a correlation between IMT and fasting LDL cholesterol in dialysis patients.

In this study, 16% (8 out of 50) of the patients group had cardiac ECG ischemic changes, while 14% (7 out of 50) had plaques in the carotid artery wall (table 6). This goes with (Pacazice *et al.*, 1996; Savage *et al.* (1998).and Mojs *et al.*, 2000). who found that there is a significant increase in plaque occurrence in hemodialysis patients.

In this study, there was a significant positive correlation between non-fasting serum cholesterol, non-fasting non-HDL and fasting LDL and cardiac ECG ischemic changes, (table 7) (P=0.018, P=0.02, P=0.04) respectively. Also there was a significant positive correlation between non-fasting serum cholesterol, non-fasting non-HDL and carotid plaques, (table 9), (P=0.032, P=0.01), respectively. There was no significant correlation between non-fasting HDL, fasting LDL and carotid plaques.

Pascazio et al. (1996) observed a large number of vascular plaques in uremic patients. They concluded that the process of advanced atherosclerosis might be started with the beginning of renal failure, they suggested that hemodialysis treatment may not be a potential factor to accelerate atherosclerosis. Finally, they concluded that the progression of atherosclerosis might be related to atherogenic factors operative before regular dialysis.

Mojs et al. (2000). Also in their study 28 HD patients found that, age was the only significant determinant of the number of plaques. He concluded that hemdialysis patients had advanced atherosclerosis in the carotid arteries compared with normal subjects.

Previous Cross-Sectional

Studies showed that in ESRD patients,

(non-HDL-C) was an independent factor associated with carotid artery intima-media thickness (Shoji *et al.*, 2000) and aortic sclerosis (Shoji *et al.*, 1998 and Shoji *et al.*, 2001).

In this study, there was a significant positive correlation between the duration of dialysis and IMT of both carotids and left femoral arteries, while there was no significant correlation between the duration of dialysis and IMT of right femoral artery. This goes with Burdick *et al.* (1994), who found a positive significant relation between carotid intima-media thickness and dialysis duration.

In contrast, correlation of IMT with ages and duration of hemodialysis in HD patients was evaluated, by Shoji and by Mojs. No clear relationship of IMT with duration of hemodialysis treatment was found in their studies (Mojs *et al.*, 2000; Shoji *et al.*, 2002).

In previous studies, the IMT of carotid arteries was not associated with HD duration, thus suggesting that the atherosclerosis may be accelerated by the uremic state per se rather than by the hemodialysis (Rawagishi et al., 1995; London *et al.*, 1996; Mojs *et al.*, 2000).

In our study, there was a positive correlation between cardiac ECG ischemic changes and IMT of both carotids and left femoral arteries, while there was no significant correlation between cardiac ECG ischemic changes and IMT of right femoral artery (table 8).

In this study, there was a significant positive correlation between carotid plaques and IMT of both carotids and left femoral arteries, (P<0.05, table 10), while there was no significant correlation between carotid plaques and IMT of right femoral artery (table 10).

These findings are in accordance with the study of Mojs *et al.* (2000), that involved 102 non-diabetic patients, B-mode ultrasonography was used to compare IMT and plaque occurrnce in the carotid arteries

of these patients with those of 30 control subjects without renal failure. The IMT was higher in the HD patients (0.75 mm versus 0.62 mm), more patients had plaques (64% versus 23%) than in the control group and the number of plaques in the patients was greater.

Mojs et al. (2000) found that IMT values also correlated with total and LDL cholesterol. Plaque occurrace correlated with age, total and LDL cholesterol, triglycerides and smoking the number of plaques correlated with age and with total and LDL cholesterol.

Non fasting non-HDL-C (total cholesterol minus HDL) is an additional target and should be less than 130 mg/dl, if triglyceride levels are greater than or equal to 200. Non-HDL cholesterol has the additional benefit that it can be measured using non-fasting blood specimens (NKF guidelines, 2003 and Nishizawa *et al.*, 2003) found that non-fasting non-HDL-C predicts future cardiovascular mortality in a cohort of hemodialysis patients.

In our study, level of non-HDL more than 130 mg/dl was observed in more than 65% f patients group versus 30% in control group. Also, there was positive correlation between nonfasting non-HDL and IMT of both carotids and both femoral arteries in patients group, therefore non-fasting non-HDL with a cut off 130 mg/dl is a good predictor of atherosclerosis in hemodialysis patients (table 5).

Conclusion

In our study, we found that non-fasting non-HDL was as relevant as fasting LDL in predictiron of occurrace of cardiovascular complications in HD patients, but it has the advantage that it can be measured non-fasting.

References

- 1. **Attman PO and Alaupovic P (1991):** Lipid and apolipoprotein profiles of uremic dyslipoproteinemia: relation to renal function and dialysis. Nephron, 57(4):401-410.
- 2. Benedetto F A, Mallamaci F, Tripepi G and Zoccali C (1991): Prognostic value of

- ultrasonographic measurement of carotid intima-media thickness in dialysis patients. J. Am. Soc. Nephrol., 12(11):2458-2464.
- Bernadette EA, Mallaci F, Tripepi G and Zoccali C (2001): Prognostic value of carotid intima-media thickness in dialysis patients. J. Am. Soc. Nephrol., 12:2458-2464.
- Burdick L, Periti M, Salvaggio A, Bertoli, S, Mangiorotti R, Castagnone D and Anguis-Sola G (1994): Relation between carotid artery atherosclerosis and time on dialysis: a non-invasive study in vivo. Clin. Nephrol., 42(2):121-126.
- Damjanovic T and Dimkovic N (2003): Dialysis as a risk factor for development of atherosclerosis. Med. Pregl., 56:17.
- 6. Mahn R, Qette K, Mondorf M, Finke K and Sieberth MG (1983): Analysis f cardiovascular risk factors in chronic hemodialysis patients with special attention to hyperlipoproteinemias Atherosclerosis, 48:279-288.
- 7. **Mojs R (2000) :** Carotid intima-media thickness and plaques in hemodialysis patients. Artif. Organs, 24(9):691-695.
- 8. Jungers P, Massy Z A, Nguzen Khoa T, Fumeron C, Labrunie M, Lacour B et al. (1997): Incidence and risk factors of atherosclerotic cardiovascular accidents in predialysis chronic renal failure patients: a prospective study. Nephrol. Dial. Transplant., 12:2597-2602.
- 9. Kato A, Takako T, Yukitata M, Miromishi K and Akvia H (2003): Impact of carotid atherosclerosis on long-term mortality in chronic hemodialysis patients. Kidney Internationa, 64:1472.
- Kawaguishi T, Nishizawa Y, Konishi T, Kawasaki K, Emoto M, Shoji T, Tabata T, Inove T and Morii H (1995): High resolution B-mode ultrasonography in evaluation of atherosclerosis in uremia. Kidney. Int., 48(3):820-826.
- 11. Koch M, Kutkuln B, Trenkwalder E et al. (1997): Apolipoprotein B., Fibrinogen, HDL cholesterol and apolipoprotein (a) phenotypes predict coronary artery disease (CAD) in hemodialysis patients. J. Am. Soc. Nephrol., 8:1889-1898.
- 12. Koniger M K, Quaschning T, Wanner C, Schollmeyer P and Kramer-Guth A (1999): Abnormalities in lipoprotein metabolism in hemodialysis patients. Kidney Int., 56(71 suppl): 248S-250S.
- 13. London GM, Guerin AP, Marchais SJ, Pannier B, Safar ME, Day M and Metivier F (1996): Cardiac and arterial

- interactions in end-stage renal disease. Kidney Int, 50(2):600-608.
- 14. Lowrie EG and Lew N L (1990): Death risk in hemodialysis patients: the predictive value of commonly measured variables and an evaluation of death rate differences between facilities. Am. J. Kidney, Dis., 15(5):458-482.
- Mittman N and Avram MM (1996): Dyslipidemia in renal disease. Seminars in nephrology; 16:202-213.
- 16. NCEP (National cholesterol education program) (2001): Executive summary of the third report of the National cholesterol education program (NCEP), expert panel on detection, evaluation and treatment of high blood cholesterol in adults (adult treatment panel III). JAMA 285:2486-2497.
- 17. National kidney foundation. K./DOQI (2003): Clinical practice guidelines for managing dyslipidemia in chronic kidney disease. Am. J. Kidney. Dis., 41:S1-92.
- Nishizawa Tetsuo Shoji Ryusuke Kakiya, Yoshihiro Tsujimoto et al.(2003): Nonhigh density lipoprotein cholesterol (non-HDL-C) as a predictor of cardiovascular mortality in patients with end-stage renal disease. Kidney Int., Vol 63; Suppl. 84:117-120.
- 19. sPapagianni A, Kalovoulos M, Krimizis D, Vainas A, Belechri AM, Alexopoulos E and Memmos D (2003): Carotid atherosclerosis is associated with inflammation and endothelial cell adhesion molecules in chronic haemodialysis patients. Nephrol. Dial. Transplant., 18:113-119.
- Pascazio L, Bianco F, Giorgini A, Galli G, Curri G and Panzetta G (1996): Echocolor Doppler imaging of carotid vessels in hemodialysis patients; evidence of high levels of atherosclerotic lesions. Am. J. Kidney. Dis., 28(5):713-720.
- 21. Rattassi M, Puato M, Faggin E, Bertipaglia B and Grego F (2003): New markers of accelerated atherosclerosis in endstage renal disease. J. Nephrol., 16:11-20
- Rostand SG, Kirk K A and Rutsky EA (1984): Dialysis-associated ischemic heart disease; Insights from coronary angiography. Kidney. Int; 25:653-659.

- 23. Sarnak M J and Levey A S(2000): Cardiovascular disease and chronic renal disease: a new paradigm. Am. J. Kidney. Dis., 35:S117-S131.
- 24. Savage T, Clarke A L, Giles M, Tomson C R V and Raine A E G (1998): Calcified plaque is common in the carotid and femoral arteries of dialysis patients without clinical vascular disease. Nephrol. Dial. Transplant., 13(8):2004-2014.
- 25. Shlipak M G, Fried L F, Crmp C et al. (2002): Cardiovascular risk status in elderly patients with renal insufficiency. Kidney. Int., 62:997-1004.
- 26. Shoji T, Nishizawa Y, Kawagishi T, Tanaka M, Kawasaki K, Tabata T, Inove. T and Morii H (1997): Atherogenic lipoprotein changes in the absence of hyperlipidemia in patients with chronics renal failure treated by hemiodialysis. Atherosclerosis, 131:229-236.
- 27. Shoji T, Nishizawa Y, Kawagishi T *et al.* (1998): Intermediate density lipoprotein as an independent risk factor for aortic atherosclerosis in hemodialysis patients. J. Am. Soc. Nephrol., 9:1277-1284.
- Shoji T, Kawagishi T, Emoto M, Mackawa K, Tan-waki H, Kanda M and Nishizawa Y (2000): Additive impacts of diabetes and renal failure on carotid atheroscerlosis. Atherosclerosis, 153:257-258.
- 29. **Shoji T, Emoto M, Shinohara K** *et al.*(2001): Diabetes mellitus, aortic stiffness and cardiovascular mortality in end-stage renal disease. Am. Soc. Nephrol., 12:2117-2124.
- 30. Shoji T, Emoto M, Tabata T, Kimoto E, Shinohara K, Mackawa K et al. (2002): Advanced atherosclerosis in predialysis patients with chronic renal failure. Kidney International; 61:2187.
- 31. Takashiro M, Simogawa O, Fukiyama, K. et al.(2001): Significant association between the progression of coronary artery calcification and dyslipidemia in patients on chronic hemodialysis. Am. J. Kidney. Dis., 2001, 38:64-69.
- 32. U.S. Renal Data System, USRDA 2003
 Annual Data Report (2003): Atlas of endstage renal disease in the united states,
 National Institutes of Health, National
 Institute of Diabetes and Digestive and
 Kidney Diseases, Bethesde, M.D.

البروتينات الدهنية غير عالية الكثافة في المرضي غير الصائمين كمؤشر لأمراض القلب وتصلب الشرايين في مرضي الفشل الكلوي المزمن المعالجين بالإستصفاء الدموي

أ.د./ صبري جوهر *، أ.م./ مني حسني * ، أ.م./ هوايدا عبد الحميد *، طبيب / ياسر ابراهيم * *

* قسم الباطنة العامة - كلية الطب - جامعة عين شمس * *معهد دمنهور الطبي

أستهدف هذا البحث الدراسة والمقارنة بين الدهون غير عالية الكثافة في المرضي غير الصائمين والدهون منخفضة الكثافة في المرضي الصائمين كمؤشر لتصلب الشرايين في مرضي الفشل الكلوي المزمن المعالجون بالإستصفاء الدموي المتكرر.

وقد شملت الدراسة خمسون مريضاً بالفشل الكلوي المزمن والمعالوجون بالإستصفاء 40.4 الدموي المتكرر (31 رجل، 19 إمراة) يتراوح أعمارهم ما بين 18 : 62 سنة بمتوسط عمر 40.4 سنة وعشرة من الأصحاء (7 رجال و 3 نساء) كحالات متابعة تتراوح أعمارهم ما بين 22 $\frac{1}{2}$ سنة بمتوسط عمر 40.4 $\frac{1}{2}$ سنة بمتوسط عمر 10.4 $\frac{1}{2}$ سنة بمتوسط عمر 10.4

وقد تم تقسيم المرضي حسب سبب الفشل الكلوي إلى : مرضي التهاب حبيبات الكلي (15 مريض)، مرضي الإلتهاب الصديدي للكلي (5 مريض)، مرضي الإلتهاب الصديدي للكلي المرتفع (17 مريض)، مرضي التهاب الكلي الناتج عن المسكنات (2 مريض)، مرضي غير معروف سبب الفشل الكلوي لديهم (10 مرضي).

كما تراوحت مدة العلاج بالإستصفاء الدموي ما بين مدد أقل من سنة إلى مدد أكثر من 5 سنوات.

وقد تم عمل الأتي لجميع المرضي والأصحاء:

أخذ تأريخ مرضي كامل لهم، فحص إكلينيكي شامل لهم، رسم قلب عادي، ودوبلكس على الشريان السباتي الأيمن والأيسر، تحاليل كيميائية وتشمل: الكولسترول والدهون عالية الكثافة في المرضي غير الصائمين والدهون منخفضة الكثافة في المرضي المرضي الصائمين.

وكانت نتائج البحث كالأتي:

- إرتفاع مستوي الدهون غير عالية الكثافة في المرضي غير الصائمين.
- سمك الطبقة الداخلية حتى الطبقة الوسطي كان أعلي بدرجة ملحوظة في مرضي الإستصفاء الدموي مقارنة بالأصحاء من نفس العمر والجنس.
- كان هناك علاقة بين سمك الطبقة الداخلية حتى الطبقة الوسطي بالشريان الثباتي ومدة الإستصفاء الدموي.
- لم يكن هناك علاقة بين سمك الطبقة الداخلية حتى سمك الطبقة الوسطي من الشريان الثباتي والشريان الفخذي ومستوي الدهون عالية الكثافة والكولسترول في المرضي غير الصائمين.
- في حين كان هناك علاقة تبين سمك الطبقة الداخلية حتى سمك الطبقة الوسطي ومستوي الدهون غير العالية الكثافة في المرضي غير الصائمين والدهون منخفضة الكثافة في المرضي المرضي الصائمين.
- 16% من المرضي وجدوا أنهم يعانون من قصور وضيق الشريان التاجي وفي هؤلاء المرضي وجد علاقة بين سمك الطبقة الداخلية حتى الطبقة الوسطي من الشريان السباتي

Sabry Gohar et al

- الأيمن والأيسر والشريان الفخذي الأيسر في حين لم يكن هناك علاقة بين سمك الطبقة الداخلية حتى سمك الطبقة المتوسطة من الشريان الفخذي الايمن.
- كان هناك علاقة بين مدة الغسيل الدموي وسمك الطبقة الداخلية حتي الطبقة الوسطي من الشريان الثباتي الأيمن والأيسر والشريان الفخذي الأيسر في حين لم تكن هناك علاقة بين سمك الطبقة الداخلية حتي الطبقة الوسطي من الشريان الفخذي الأيمن ومدة الغسيل الكلوي.